

Blockade of hyperpolarization-activated channels modifies the effect of β -adrenoceptor stimulation

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Abstract

Experiments on rats showed that blockade of hyperpolarization-activated currents moderates tachycardia induced by β -adrenoceptor agonist isoproterenol and potentiates the increase in stroke volume produced by this agonist. Electrical stimulation of the vagus nerve against the background of isoproterenol treatment augmented bradycardia and increased stroke volume. Blockade of hyperpolarization-activated currents followed by application of isoproterenol moderated vagus-induced bradycardia and had no effect on the dynamics of stroke volume.

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Keywords

Heart, Hyperpolarization currents, Rat, Sympathetic influences, Vagus